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EXAMINER

CHONG, KIMBERLY

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1635

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PAPER

**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.



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**BEFORE THE BOARD OF PATENT APPEALS  
AND INTERFERENCES**

Application Number: 09/993,183  
Filing Date: November 14, 2001  
Appellant(s): GEWIRTZ, ALAN

\_\_\_\_\_  
George A. Frank  
For Appellant

**EXAMINER'S ANSWER**

This is in response to the appeal brief filed November 5, 2007 appealing from the Office action mailed March 30, 2007.

**(1) Real Party in Interest**

A statement identifying by name the real party in interest is contained in the brief.

**(2) Related Appeals and Interferences**

The examiner is not aware of any related appeals, interferences, or judicial proceedings which will directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal.

**(3) Status of Claims**

The statement of the status of claims contained in the brief is correct.

**(4) Status of Amendments After Final**

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

**(5) Summary of Claimed Subject Matter**

The summary of claimed subject matter contained in the brief is correct.

**(6) Grounds of Rejection to be Reviewed on Appeal**

The appellant's statement of the grounds of rejection to be reviewed on appeal is substantially correct. However, because the Kreutzer et al. document (WO 00/44895) is

not available as prior art because it has been previously antedated, there is a new 35 U.S.C. 103 rejection which is very similar to the rejection of record because the Kreutzer et al. document was relied upon as evidentiary support.

#### **GROUND OF REJECTION NOT ON REVIEW**

The following grounds of rejection have not been withdrawn by the examiner, but they are not under review on appeal because they have not been presented for review in the appellant's brief. Claims 30-31 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement.

#### **(7) Claims Appendix**

The copy of the appealed claims contained in the Appendix to the brief is correct.

#### **(8) Evidence Relied Upon**

Fire et al. (US 6,506,559)

Gewirtz et al. (WO 92/19252)

Sharp et al. (Genes and Development 13: 139-141, 1999)

Kreutzer et al. (WO 00/44895)

#### **(9) Grounds of Rejection**

The following ground(s) of rejection are applicable to the appealed claims:

The following includes a new ground of rejection because the Kreutzer et al. document is not available as prior art because it has previously been antedated by the declaration filed 04/28/2006. Although the rejection is new, the rejection is essentially the same as the previous rejection because the Kreutzer et al. document was only relied upon as evidentiary support for the fact that the method of RNAi in human cells taught by Fire et al. reference was enabled and Kreutzer et al. reduced to practice the methods taught by Fire et al.

***Claim Rejections - 35 USC § 102***

Claims 1, 2, 5, 7-9, 11, 21-22, and 24-27 are rejected under 35 U.S.C. 102(e) as being anticipated by Fire et al. (U.S. Patent 6,506,559).

Fire et al. disclose a method for inhibiting expression of a target gene using double stranded RNA to induce RNAi in a cell *in vitro* (Column 26, claim 1) wherein the cell is from an animal (Column 26, claim 6). Fire et al. disclose that the cell with the target gene may be derived from or contained in any organism (column 8, line 13-14) and that examples of vertebrate animals include mammals and human (column 8, lines 35-37) and that the cell having the target gene may be "immortalized or transformed, or the like" (column 8, lines 52-55) and that "the present invention could be used for treatment or development of treatments for cancers of any type, including solid tumors and leukemias..." (Column 10, lines 26-28). Fire et al. disclose that lipid mediated carrier transport can be used to introduce nucleic acids to cells (Column 9, lines 55-60). Fire et al. also disclose that inhibition of gene expression refers to the absence (or

observable decrease) in the level of protein and/or mRNA product from a target gene (Column 6, lines 55-57), thereby indicating disruption of gene function (which is to produce protein).

Therefore, Fire et al. anticipate the instant invention as set forth in claims 1, 2, 5, 7-9, 11 and 21-22 and 24-27.

***New Claim Rejections - 35 USC § 103***

Claims 1, 2, 5, 7-9, 11 and 21-38 are rejected under 35 U.S.C. 103(a) as being unpatentable over Fire et al. (US Patent No. 6,506,559) as applied to claims 1, 2, 5, 7-9, 11, 21-22 and 24-27 in the 102(e), Gewirtz et al (WO 9219252) and Sharp (Genes and Development 1991, 13: 139-141).

Fire et al. teach a method for inhibiting expression of a target gene using double stranded RNA to induce RNAi in a cell *in vitro* (Column 26, claim 1) wherein the cell is from an animal (Column 26, claim 6) and the dsRNA has a length of less than about 830 bp (see Table 1). Fire et al. teach that the cell with the target gene may be derived from or contained in any organism (column 8, line 13-14) and that examples of vertebrate animals include mammals and human (column 8, lines 35-37) and that the cell having the target gene may be "immortalized or transformed, or the like" (column 8, lines 52-55) and that "the present invention could be used for treatment or development of treatments for cancers of any type, including solid tumors, sarcomas and leukemias..." (Column 10, lines 26-28). It must be noted that the limitation "selecting a human cell expressing the target gene" is not defined in the specification, so for prior art

purposes, this recitation is being interpreted to mean a cell line that contains a target gene and is capable of being treating with a dsRNA and is therefore anticipated by Fire et al. Fire et al. teach target genes that are oncogenes (col. 11). Fire et al. teach that lipid mediated carrier transport can be used to introduce nucleic acids to cells (Column 9, lines 55-60). Fire et al. also teach that inhibition of gene expression refers to the absence (or observable decrease) in the level of protein and/or mRNA product from a target gene as determined by measurement of the target gene or expression from said target gene (Column 6, lines 55-57), thereby indicating disruption of gene function (which is to produce protein). Fire et al. teach that using the methods of their invention, gene disruptions may be used to discover the function of a target gene and to produce disease models in which the target gene is involved in causing or preventing a pathological condition (col. 5, lines 30-37). Fire et al. disclose that relative to antisense approaches, their invention has advantages in the stability of the material to be delivered (col. 3, line 20).

Fire et al. do not teach the nucleotide sequence of the oncogene c-Kit.

Gewirtz et al. teach the antisense inhibition of c-Kit proto-oncogene expression in human cells and that c-kit antisense oligonucleotides are particularly useful against leukemia and melanoma (see page 15, lines 6-25). Gewirtz et al. disclose that the c-Kit cDNA sequence was known in 1987 and cite Yarden et al. Gewirtz et al. do not specifically teach inhibition in HL-60 cell lines or CHP 100 cell lines. Collin et al. teach HL-60 leukemic cells lines provide a unique and efficient in vitro cell model to study the cellular and molecular events involved in the progression of leukemia (see Abstract

page 1233). Likewise Pence et al. teach the CHP 100 neuroblastoma cell line is useful in studying the progression of neuroblastoma in patients.

Sharp is added as a general reference supporting the idea that RNAi is a general mechanism that is likely to be a general mechanism for gene regulation and may be critical for many developmental and antiviral processes.

It would have been *prima facie* obvious to one of ordinary skill in the art, at the time the instant invention was made, to substitute an dsRNA oligonucleotide in place of the antisense oligonucleotide in a method of inhibiting the expression of the oncogene c-Kit *in vitro* using an antisense inhibitor in human leukemia cells (as taught by Gewirtz et al.), wherein the dsRNA was comprised in pharmaceutical composition (as taught by Fire) because antisense inhibition of c-Kit was taught in the prior art as inhibiting the expression of KitR in human leukemia cells (as taught by Gewirtz et al.), because dsRNA can be used to initiate RNA interference *in vitro* by targeting oncogenes in human cells including leukemia (as taught by Fire) and because relative to antisense approaches, dsRNA used to inhibit gene expression has advantages in the stability of the material to be delivered (as taught by Fire).

It would have been further obvious to use a HL-60 cell line for the study of leukemia *in vitro* and further obvious to use CHP 100 to study the cellular events associated with neuroblastoma. Fire et al. does not specifically disclose the optimal time of incubation of said dsRNA with a cell or the optimal concentration of dsRNA used but it would have been obvious to one of skill in the art and a matter of routine



optimization to determine the amount of time to expose the dsRNA to the cell to achieve the most efficient gene interference and to determine the optimal workable ranges of a dsRNA that most efficiently caused gene interference in a cell. MPEP 2144.05 states in part "where the general conditions of a claim are disclosed in the prior art, it is not inventive to discover the optimum or workable ranges by routine experimentation."

One of ordinary skill in the art would have been motivated to practice a method of inhibiting the expression of the oncogene c-Kit *in vitro* in human leukemia cells or melanoma cells (as taught by Gewirtz et al.) using a dsRNA to initiate RNA interference wherein the dsRNA was comprised in pharmaceutical composition (as taught by Fire) because antisense inhibition of c-Kit was taught in the prior art as inhibiting the expression of KitR in human leukemia cells (as taught by Gewirtz et al.) and because relative to antisense approaches, dsRNA used to inhibit gene expression has advantages in the stability of the material to be delivered and has advantages of sequence specificity (as taught by Fire et al.).

One of ordinary skill in the art would have expected success in practicing a method of inhibiting the expression of the oncogene c-Kit *in vitro* in human leukemia cells (as taught by Gewirtz et al.) using a dsRNA to initiate RNA interference wherein the dsRNA was comprised in pharmaceutical composition (as taught by Fire) because antisense inhibition of c-Kit was taught in the prior art as inhibiting the expression of KitR in human leukemia cells (as taught by Gewirtz et al.), because Fire et al. teach that dsRNA can be used to initiate RNA interference in human cells and because relative to

antisense approaches, dsRNA used to inhibit gene expression has advantages in the stability of the material to be delivered (as taught by Fire). Moreover, one would have had a reasonable expectation of success at initiating RNA interference in human cells because Sharp further supports the fact that RNAi is a general mechanism that is likely to be a general mechanism for gene regulation and may be critical for many developmental and antiviral processes.

Therefore, the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

#### **(10) Response to Argument**

Appellants submit the claims are novel over Fire et al. because Fire et al. is not enabled. Appellants cite several court decisions to outline the legal standard for anticipation. Appellants argue the cited portions of the Fire et al. reference relied upon in the rejection when viewed in the context of the surrounding disclosure, are vague and general representations of the claimed invention and are not sufficient to support a rejection of anticipation. Appellants submit Fire et al. generally describes a dsRNA inhibition phenomenon in invertebrate animals such as the worm but this teaching does not enable the practice of inhibition in mammalian cells, as instantly claimed.

Appellants attempt to summarize the state of the art from the time of filing of Fire et al. up through the time of Appellant's filing as evidence that Fire et al. did not enable the practice of the claimed invention in any mammal, including humans.

It is not disputed that Fire et al. did not exemplify his invention in human cells, but just because Fire et al. did not reduce to practice his invention does not mean the invention was not enabled. Since the issuance of the Fire et al. patent (the '559 patent), which is presumed to be enabled, thousands of post-filing art references have repeatedly shown that the methods of Fire et al. work in human cells. In fact, Fire et al. won a Nobel Prize for their discovery, largely based upon the implications of its use in humans, and the discovery that the process underlies an entire RNA-dependent system of gene regulation that is conserved at some level across virtually every multi-cellular organism (see Zamore, Nat. Struct. Biol. From the IDS for review). Because Appellants have not shown any manipulative differences or shown any structural differences in the steps used in the instantly disclosed methods as compared to the methods disclosed by Fire et al., Appellant's position that Fire et al. does not teach or does not enable a skilled artisan to practice each and every limitation of the claimed invention is not convincing.

Appellants cite Fire's own work published in Trends in Genetics 1999 (Exhibit I, Evidence Appendix) to establish that Fire himself had no knowledge that RNAi could be practiced in mammals and "did not believe the simple protocols used for invertebrates and plants would be successful in mammalian or human cells." Appellants cite a passage from the reference for the supposed belief by Fire and summarize this as an admission by Fire that his invention in the '559 patent was "limited to the invertebrate animals". The paragraph cited by Appellant is not evidence that Fire felt his invention was limited to invertebrate animals and his thoughts on whether this would work was

mere speculation and was not an admission by Fire as alleged by Appellant. To the contrary, this mere speculation has proved to be unfounded given the voluminous amount of post-filing art that has shown the methods of Fire et al. work in human cells.

Appellants argue that Fire summarizes the state of the art at that time and teach that if RNAi existed in cells, it would probably be necessary to induce a transient lapse in PKR response or use a dose incapable of activating the PKR response to practice RNAi in mammalian cells, yet Fire et al. provides not guidance on any methods or means of inducing a lapse in the PKR response nor were any methods predictable at the time of Appellants filing. Appellants arguments are not convincing because there are no manipulative steps taken by Appellant that Fire did not do that serves to provide any guidance on a means of inducing a lapse in the PKR response. Further, although Appellants have exemplified RNAi in mammalian cells (see page 12), this was done using an 828 nucleotide base pair dsRNA, a long dsRNA used by Fire et al. exemplified to mediate RNAi in invertebrates and taught to be capable of mediating RNAi in human cells. Therefore, Appellants have merely reduced to practice the invention taught by Fire et al., an invention that was, therefore, enabled.

Appellants argue the statement cited by Examiner in Sharp et al. was taken out of context and because the entire paper is focused on invertebrates and because Sharp et al. suggested that RNAi was an established phenomenon in invertebrates, the skilled artisan could not have practiced Appellant's claimed invention based on the disclosure of Fire and the state of knowledge that existed at that time. Appellants are correct in stating that Sharp et al. primarily discusses RNAi in invertebrates, but the statement by

Sharp et al. that RNAi may perhaps occur or can be induced in mammalian cells would provide the motivation to one of skill in the art, as indicated in the 103 rejection of record, to reduce the invention, taught by Fire et al., to practice in mammalian cells.

Appellants cite Paddison et al., Wianny et al. and Svoboda et al. (Exhibits, 5, 6 and 7, respectively) for the notion that RNAi in mammalian cells had only been accomplished in early embryonic cells of the mouse and states these cells are not directly relevant to the practice of RNAi in mammalian cells generally because they did not solve the problem regarding the practice of RNAi in developed cells i.e. cells having a PKR response. This argument is not convincing for several reasons. First, neither Paddison et al., Wianny et al. nor Svoboda et al. alludes to the fact that the embryonic cells of the mouse lack the PKR response and that the RNAi seen in these cells is not representative of mammalian cells in general. Secondly, there is no distinction made in the Fire et al. reference to embryonic versus developed mammalian cells and the PKR response or lack thereof in one type of mammalian cells versus another. Likewise, there is no distinction made in the instantly claimed invention to embryonic versus developed mammalian cells and the PKR response or lack thereof. Lastly, the instant claims are drawn to disrupting expression of a gene in a mammalian cell and no distinction is made as to what developmental stage the cell is in and whether or not the cell is capable of mounting a PKR response to dsRNA. Thus, Paddison et al., Wianny et al. and Svoboda et al. in fact support the methods of Fire et al. i.e. they reduced to practice the invention taught by Fire et al. of RNAi in mammalian cells.

Appellants submit the declaration submitted by Dr. Alan Gewirtz provides evidence that a skill artisan would not have been able to practice the claimed invention based on Fire et al. and the knowledge in the art. As stated previously and reiterated herein, this assertions provided by Appellant are mere speculation that the methods taught by Fire et al. would not work in mammalian cells and does not in any way indicate undue experimentation would be necessary to practice the methods taught by Fire et al. In fact, this mere speculation has proved to be unfounded given the voluminous post-filing art that has shown the methods of Fire et al. work in human cells. As such, these assertions are not considered sufficient to consider the presumably valid patent of Fire et al. to not be enabled.

Appellants further argue that the Examiner has applied an incorrect legal standard for determining enablement of the Fire et al. reference. Appellants submit that while the Examiner may have correctly observed that the legal standard for enablement under 35 U.S.C. §102 is different than under 35 U.S.C. §112 as cited in Impax Labs, Inc. v. Aventis Pharm (Fed. Cir. 2006) (hereinafter Impax), the cited language from Impax does not provide an adequate legal standard for the determination in this instance because the reliance on language in Impax distinguishing "making" from "using" is misplaced. Appellants assert that Fire taught neither how to "make" nor how to "use" methods of RNAi in vertebrate systems and Fire et al. would not have enabled the skilled artisan to practice the claimed invention. Appellants further assert that the proper issue in Impax was whether the prior art reference describes the invention sufficiently to enable a person of ordinary skill in the art how to carry out the invention.

Appellants assert the proper considerations of enablement of a prior art reference requires factual analysis under *In re Wands* to determine whether a skilled artisan could practice the claimed invention without undue experimentation and argues *Fire et al.* fails to meet the standards of enablement as required in *In re Wands* and *In re Goodman*.

As stated in the Office action mailed 03/30/2007 and reiterated herein, the decision by the Court in *In re Goodman* is not applicable to the instant application because the legal standard for enablement under 35 U.S.C. §102 is different than under 35 U.S.C. §112. The Court in *Impax* clarified the legal standard for a prior art reference to be anticipatory. The Court stated "...anticipation does not require actual performance of suggestions in a disclosure. Rather, anticipation only requires that those suggestions be enabled to one of skill in the art." The Court further stated that "[w]hether a prior art reference is enabling is a question of law based upon underlying factual findings." The Court concluded that a "102 prior art reference does not have to be 'effective' to be enabling and thus anticipating" and the proper issue to consider upon deciding whether or not a prior art reference is enabling is if "it describes the claimed invention sufficiently to enable a person of ordinary skill in the art to carry out the invention."

*Fire et al.* recognized that double stranded RNA-mediated inhibition has advantages both in stability of the material to be delivered to the cells and the concentration required for effectiveness and further the double stranded RNA were capable of inhibiting gene expression of a target gene in a cell in vitro from an animal. *Fire et al.* teach the cell with the target gene may be derived from or contained in any organism, such as mammalian cells. *Fire et al.* recognized the methods of their

invention may be used to discover the function of a target gene involved in causing or preventing a pathological condition. Fire et al. teach general concentrations of dsRNA and routes of administration to use and although Fire et al. does not provide evidence that dsRNA would be effective in cells such as mammalian cells, Fire et al. is enabling because it describes the claimed method of mediating RNA interference in any cell type, specifically mammalian cells, sufficiently enough to enable a person of ordinary skill in the art to carry out the invention. Further evidence that Fire et al. sufficiently described methods of mediating RNAi in human cells comes from Appellants instantly claimed invention because there are not any manipulative differences or any structural differences in the steps used in the instantly disclosed methods as compared to the methods disclosed by Fire et al. Appellants have merely reduced to practice the claimed invention taught by Fire et al. Even further evidence is provided by the voluminous post-filing art that have shown the methods of Fire et al. work in human cells, in particular as evidenced by Kreutzer et al. (Reference AF, Appellants IDS filed 10/03/2004) who has shown RNAi in mammalian murine cells, cells which one of skill in the art would nonetheless recognize are generally representative of human cells in the absence of specific evidence to the contrary.

Thus, Fire et al. anticipates the instantly claimed invention.

Appellants further submit the claims are patentable over Fire et al. in view of Kreutzer et al., Gewirtz and Sharp. As stated above Kreutzer et al. is not available as prior art because it has previously been antedated by the declaration filed 04/28/2006, however the claims remain rejected over Fire et al., Gewirtz et al. and Sharp et al.,



therefore response to Appellants arguments regarding Kreutzer et al. are obviated. Appellants rely on arguments of Fire et al. above and further assert there would be no motivation to combine the references of Fire et al. with Gewirtz et al. and Sharp because at the time of filing, the prior art did not know whether mechanisms for RNAi existed or could be leveraged for gene silencing in vertebrate, mammalian and especially human cells. Appellants assert even if the references were combinable, there would have been no expectation of success because the skilled artisan did not know whether RNAi could be induced for a particular gene in mammalian cells. Appellants further assert the references, even in combination, do not disclose each and every feature of the claimed invention because it the method of disrupting gene expression is not taught "in a human cell" and further assert the prior art teaches away from the claimed invention because the state of the art was "fraught with uncertainty and unpredictability with respect to the application of RNAi in mammalian systems" and the art as a whole, including Fire et al., taught away from the claimed invention. Appellants assert that Gewirtz et al. while teaching treatment of human cells with an antisense compound targeted to c-kit RNA, does not teach RNAi in human cells or in any cells and Gewirtz does not provide the information lacking in Fire to practice the a method of mediating gene silencing using dsRNA in human cells.

The response to Fire et al. is relied upon as above. Appellants assertion that one of skill would not have been motivated to combine the references because the prior art proves the uncertainty of whether mechanisms for RNAi existed in mammalian cells is not convincing. The few references cited by Appellant, namely Sharp, Paddison et al.,

Wianny et al. and Svoboda et al., do not prove one of skill in the art would not have been motivated to practice the methods of Fire et al. in mammalian cells. In fact, as discussed above, each of the references provide motivation to use the RNAi methods taught by Fire et al. to reduce the expression a c-kit RNA taught by Gewirtz. As stated above, that statement by Sharp that RNAi may perhaps occur or can be induced in mammalian cells would provide the motivation to one of skill in the art to substitute the antisense of Gewirtz with the dsRNA taught by Fire et al. to mediate gene silencing of c-kit RNA in human leukemia cells. Moreover, given that fact that Paddison et al., Wianny et al. and Svoboda et al. support the methods of Fire et al. because they reduced to practice the invention taught by Fire et al. of RNAi in mammalian cells, this is indicative that the prior art was not uncertain and unpredictable with respect to the application of RNAi in mammalian cells. Thus Appellants assertion that one of skill in the art would not have been motivated given the uncertainty of the prior art with respect to RNAi in mammalian cells is unsupported and the claimed invention would have been obvious to one of skill in the art.

Appellant's assertion that one of skill in the art would not have had a reasonable expectation of success is based on their argument that because Fire et al. did not teach RNAi in cells and the prior art was uncertain of the RNAi mechanisms in human cells, one of skill in the art would not have expected success at initiating RNAi in mammalian cells. This argument is not convincing because one would have expected success at practicing the methods of silencing gene expression in human leukemia cells, as taught by Gewirtz, given Gewirtz teach inhibition of gene expression using an inhibitory

antisense nucleic acid molecule and given Fire et al. teach dsRNA can be used to inhibit RNAi in cells and teach that relative to antisense approaches, dsRNA has advantages in stability and material to be delivered.

Examiner's response above explaining that Fire et al. is an anticipatory reference is relied upon in response to Appellants assertion that the combination of references does not teach all of the claimed limitation because RNAi in human cells is not taught. Fire et al. is an enabling reference and therefore is anticipatory and therefore teaches the claimed limitation of RNAi in human cells.

In response to Appellants assertion that the state of the art teaches away from the claimed invention, the references discussed by Appellant are not enough evidence to conclusively make the statement that the state of the art was "fraught with uncertainty and unpredictability with respect to the application of RNAi in mammalian systems." As stated above, the state of the art provided one of skill in the art motivation to use the dsRNA taught by Fire et al. in the methods of inhibiting c-kit RNA expression as taught by Gewirtz. Further, nowhere in the Fire et al. reference does it teach away from using dsRNA in human cells.

Thus in the absence of evidence to the contrary, the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

This examiner's answer contains a new ground of rejection set forth in section (9) above. Accordingly, appellant must within **TWO MONTHS** from the date of this answer exercise one of the following two options to avoid *sua sponte* **dismissal of the appeal** as to the claims subject to the new ground of rejection:

(1) **Reopen prosecution.** Request that prosecution be reopened before the primary examiner by filing a reply under 37 CFR 1.111 with or without amendment, affidavit or other evidence. Any amendment, affidavit or other evidence must be relevant to the new grounds of rejection. A request that complies with 37 CFR 41.39(b)(1) will be entered and considered. Any request that prosecution be reopened will be treated as a request to withdraw the appeal.

(2) **Maintain appeal.** Request that the appeal be maintained by filing a reply brief as set forth in 37 CFR 41.41. Such a reply brief must address each new ground of rejection as set forth in 37 CFR 41.37(c)(1)(vii) and should be in compliance with the other requirements of 37 CFR 41.37(c). If a reply brief filed pursuant to 37 CFR 41.39(b)(2) is accompanied by any amendment, affidavit or other evidence, it shall be treated as a request that prosecution be reopened before the primary examiner under 37 CFR 41.39(b)(1).

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Extensions of time under 37 CFR 1.136(a) are not applicable to the TWO MONTH time period set forth above. See 37 CFR 1.136(b) for extensions of time to reply for patent applications and 37 CFR 1.550(c) for extensions of time to reply for ex parte reexamination proceedings.

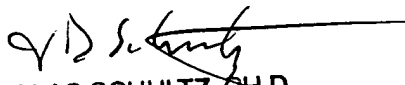
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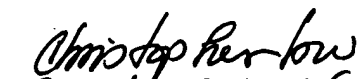
/Kimberly Chong/ Examiner AU 1635

**A Technology Center Director or designee must personally approve the new ground(s) of rejection set forth in section (9) above by signing below:**

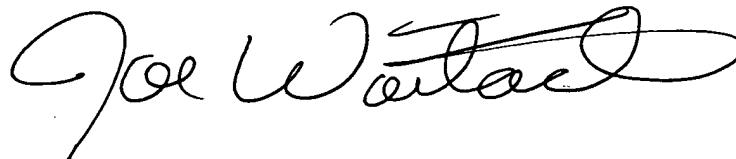
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SUPERVISORY PATENT EXAMINER

  
Director TC1600 (acting)

Joseph Woitach, SPE 1633



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